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Arm swing in human walking: What is their drive?

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ABSTRACT

Although previous research has studied arm swing during walking, to date, it remains unclear what the contribution of passive dynamics versus active muscle control to arm swing is.

In this study, we measured arm swing kinematics with 3D-motion analysis. We used a musculoskeletal model in OpenSim and generated dynamic simulations of walking with and without upper limb muscle excitations. We then compared arm swing amplitude and relative phase during both simulations to verify the extent to which passive dynamics contribute to arm swing.

The results confirm that passive dynamics are partly responsible for arm swing during walking. However, without muscle activity, passive swing amplitude and relative phase decrease significantly (both $p < 0.05$), the latter inducing a more in-phase swing pattern of the arms. Therefore, we conclude that muscle activity is needed to increase arm swing amplitude and modify relative phase during human walking to obtain an out-phase movement relative to the legs.

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1. Introduction

During walking the arms swing out of phase relative to the legs, to minimize the body's angular momentum around the vertical axis, which reduces energy expenditure [1–5].

With respect to the effect of arm swing on gait stability there is less consensus; Ortega et al. [4] found an increase in stability due to arm swing, whereas Bruijn et al. [6] and Pijnappels et al. [7] found negative effects of arm swing on gait stability.

A number of studies have focused on the "how" of this movement pattern [3,5,8–12]. In these studies, the arms are often represented as pendula [5,8–11], or as mass dampers [11] that swing passively due to thorax movements [5,8–11]. However, experimental studies using surface EMG have shown that arm swing is at least in part controlled actively [3,8,9,11–13]. Fernandez-Ballesteros et al. [12] were the first to document muscle activity in arm muscles during walking. They showed that the posterior and middle parts of the deltoid were active at contralateral heel strike, when the arm changed direction.

Barthelemy & Nielsen [13] and Kuhtz-Buschbeck & Jing [3] reported shoulder muscle activity during walking suggesting that muscle activity might be used to initiate direction changes of the arms, to keep them swinging out-of-phase with the legs. A recent review by Meyns et al. [14] concluded that it is still unclear to what extent muscle control or passive dynamics (e.g., accelerations of the thorax) determine arm swing. It could be that muscle activity merely amplifies arm swing, without changing the movement pattern qualitatively. Alternatively, muscle activity may be necessary to maintain the out of phase relationship between the arms [1,2,5,15].

Answering the question whether arm muscle activity is needed to maintain the out of phase relationship with the legs, requires analyzing how the arms would swing without muscle activity, but with passive muscle characteristics present. Modelling provides a platform that potentially can offer such insights, since it allows altering the excitations of the upper limb muscles to evaluate the effect on the kinematics. Indeed, Jackson et al. [8] and Kubo et al. [10] excluded all arm muscle activity from the gait simulations with their pendulum models, but still accounted for passive muscle characteristics. Jackson et al. [8] found a 'very small and ragged' arm movement when muscle activity was excluded from the simulation. Kubo et al. [10] did not focus on passive arm swing kinematics, but studied the transition from 2:1 to 1:1 with respect to arm to leg swing ratio. However, they hypothesized that muscle

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activity was needed to change passive arm swing amplitude and/or relative phase. In the previous studies [8,10], muscle parameters were based on mathematical equations and not on physiological values. Furthermore, these studies used simplified kinematics as input to their simulations (i.e., only the first harmonics, and thus, the accelerations at stride and step frequency). In the present study, we compare simulations with and without arm muscle excitations for normal (out-of-phase) walking using a complex musculoskeletal model that accounts for the physiological active and passive properties of all relevant muscular structures around the shoulder. Furthermore, we used experimentally collected 3D kinematics of whole body movement and ground reaction forces at different speeds as input for the simulations. These points are innovative compared to previous work and will allow us to further the current understanding on how arm swing is organized. In turn, a better insight in the organization of arm swing kinematics might improve rehabilitation techniques for patients with impairments in arm swing during walking (e.g., in stroke or cerebral palsy) [14].

2. Methods

2.1. Subjects

Five subjects (age 28.6 ± 2.61 (mean \pm SD)) participated in this study, approved by the ethical advisory board of KU Leuven. All subjects gave written informed consent. All subjects were familiar with treadmill walking, had normal or corrected to normal vision and no known neuromuscular disorders and were naïve to the specific research question.

2.2. Measurement protocol

While walking on a treadmill (custom-built, Forcelink, Culemborg, The Netherlands) three different walking speeds were imposed (0.56 m/s, 1.11 m/s and 1.67 m/s). Each condition lasted approximately 60 s, with data being collected during the last 30 s.

The three walking speeds were randomized. 3D marker data were collected using a 10 camera Vicon system (Nexus 1.7.1, Vicon-UK, Oxford, UK) sampled at 100 samples/second. Reflective markers were attached over the bony landmarks according to the full body Plug-in-Gait model. Muscle activity of both left and right shoulder anteflexors (anterior deltoid, biceps brachii) and retroflexors (posterior deltoid, triceps and latissimus dorsi) was measured using surface EMG (Zerowire, Aurion, Milan, Italy) at 1000 samples/second. The EMG electrodes were applied according to Konrad [16]. Ground reaction force data under each foot was

measured at 1000 samples/second, using force plates embedded in the treadmill.

2.3. EMG processing

The raw EMG data were corrected for offset and filtered with a dual-pass 4th order Butterworth band-stop filter between 49 and 51 Hz. Next, the data were filtered with a dual-pass 1st order high-pass filter at 20 Hz and rectified, followed by a dual-pass 4th order low-pass Butterworth filter at 10 Hz. For analyses, EMG's were time normalized to the gait cycle, and the mean of fifteen gait cycles was calculated.

2.4. Dynamic simulations

We generated muscle-driven simulations of walking in OpenSim based on experimental motion capture data. For each subject we generated one simulation per walking speed for the two conditions: with and without arm muscle activity, resulting in six simulations per subject. Initial marker data processing was performed in Nexus. Custom code in Matlab generated the appropriate file format for analysis in OpenSim.

We used the upper and lower body model from the ULB-project [17–22], but adjusted the model in order to decrease the simulation runtime. The final (adjusted) model contained 35 degrees of freedom as well as the description of the geometry and force generating capacity of 102 muscle-tendon actuators, 48 of which controlled the action of the upper limbs. For information regarding the DOF of the model we refer to the OpenSim website (https://simtk.org/home/ulb_project). In our model, we locked four DOF: the subtalar angle, the MTP angle, wrist flexion and wrist deviation. We ran muscle-generated simulations with and without arm muscle excitations according to the workflow described in Fig. 1. In a first simulation set, we calculated the muscle excitations that tracked the measured kinematics of all degrees of freedom in the model, including the arms, allowing muscle excitations to vary between 0.01 and 1. In these simulations (ACT), arm kinematics were therefore controlled by active muscle force. During the second set of simulations (PAS), upper limb kinematics were no longer tracked and the upper limb muscle excitations were limited to 0.02. The limit of 0.02, instead of 0, was introduced for numerical reasons. Although this can result in a very small level of muscle activity, it guarantees that mainly passive muscle structures influenced the arm kinematics that were induced passively through the accelerations of the neighboring segments.

The validity of the simulations was evaluated by visually comparing (1) the calculated muscle excitations during the first set

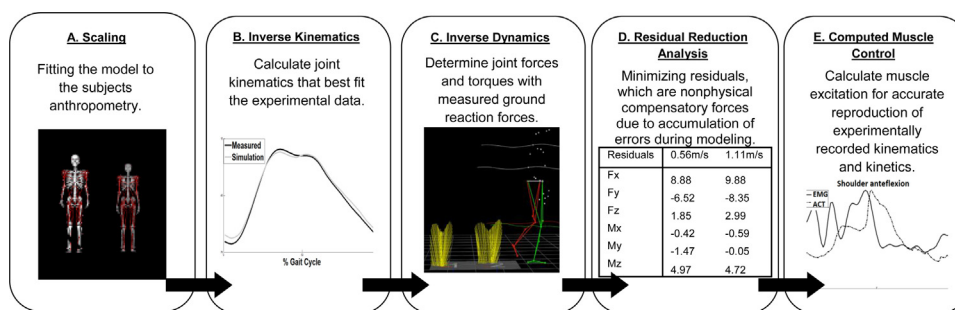


Fig. 1. Workflow of the simulation process.

First the model was scaled to the subject's anthropometry using experimentally measured marker positions (A). The Inverse Kinematics tool was then used to calculate joint kinematics that best fit the experimental marker data (B). The measured ground reaction forces were used to determine net forces and torques for each joint via the Inverse Dynamics tool (C). During the process of modelling, accumulation of errors can lead to nonphysical compensatory forces. The Residual Reduction Algorithm tool was used to minimize these residuals (D). Thereafter, the Computed Muscle Control tool (CMC) calculated muscle excitations of all muscle-tendon actuators in the model allowing accurate reproduction of the experimentally recorded kinematics and kinetics for all the experimentally measured ground reaction forces and marker data in the different conditions (E).

of simulations to the experimental EMG of the grouped synergistic actuators (Fig. 2) and (2) by comparing the tracked kinematics and kinetics of the Residual Reduction Algorithm (RRA) step to the tracked kinematics and kinetics of the Computed Muscle Control (CMC) step (Table 1). Finally, magnitudes of the average residuals during RRA were evaluated (Table 1), which were all within the determined boundaries [23].

In a post-processing step, the analysis tool was used to calculate the hand trajectory for all simulations. Arm swing amplitude was determined (from the hand trajectory data) for the first simulated gait cycle from right to second left heel strike. Relative phase between arm movements was then calculated with a custom-made Matlab script.

To determine the contribution of the passive muscle characteristics on arm swing we compared amplitude and relative phase of arms swing of simulations with (ACT) and without (PAS) muscle excitations at the three different walking speeds.

2.5. Statistics

We ran two repeated measures ANOVA's to determine the effects walking speeds (0.56 m/s, 1.11 m/s and 1.67 m/s) and simulation mode (ACT versus PAS) on: (1) arm swing amplitude and (2) relative phase. The assumption of sphericity was checked and if the Greenhouse–Geisser epsilon was ≥ 0.75 , the Huynfeldt correction was used, otherwise the Greenhouse–Geisser correction was used [24].

3. Results

Fig. 3A shows the average result with variability for the normal walking condition at 1.67 m/s for the two simulations (ACT & PAS).

3.1. Arm swing amplitude

When muscle excitations were excluded from the simulation (PAS), there was a significant decrease in arm swing amplitude

(main effect of mode of simulation, $F(1,3) = 71.57$, $p < 0.05$, Fig. 3B). When both simulations (ACT & PAS) are combined, arm swing amplitude significantly increased with walking speed (main effect of speed, $F(1.4, 4.1) = 24.22$, $p < 0.05$). Post hoc tests revealed that swing amplitude increased significantly when walking speed increased from 0.56 m/s to 1.67 m/s ($p < 0.05$) and from 1.11 m/s to 1.67 m/s ($p < 0.05$), but not from 0.56 m/s to 1.11 m/s. Moreover, arm swing amplitude showed a significant interaction between speed and simulation mode (simulation mode \times speed $F(1.27, 3.81) = 62.98$, $p < 0.05$), indicating that arm swing amplitude in the simulation with muscle excitations (ACT) increased when walking speed increased, whereas arm swing amplitude for the simulation without muscle excitation (PAS) remained similar (Fig. 3B).

3.2. Relative phase

When there were no muscle excitations present (PAS), relative phase was significantly lower (main effect of mode of simulation, $F(1,3) = 38.58$, $p < 0.05$, Fig. 3C). For both simulations (ACT & PAS), relative phase increased significantly with increasing walking speed (main effect of speed, $F(1.09, 3.27) = 10.24$, $p < 0.05$). Post hoc tests revealed that relative phase significantly increased from 0.56 m/s to 1.67 m/s ($p < 0.05$), but not from 0.56 m/s to 1.11 m/s or 1.11 m/s to 1.67 m/s. No interaction effect was found for speed and simulation mode, indicating that speed effects on relative phase were not significantly altered between the two simulations (ACT & PAS, Fig. 3C).

4. Discussion

To date, it remains unclear to what extent arm swing is actively or passively controlled. To answer this question, we used muscle driven simulations of gait in which we respectively included and excluded arm muscle excitations so that we could determine to what extent passive dynamics (induced by motions of the thorax and lower limbs) in combination with

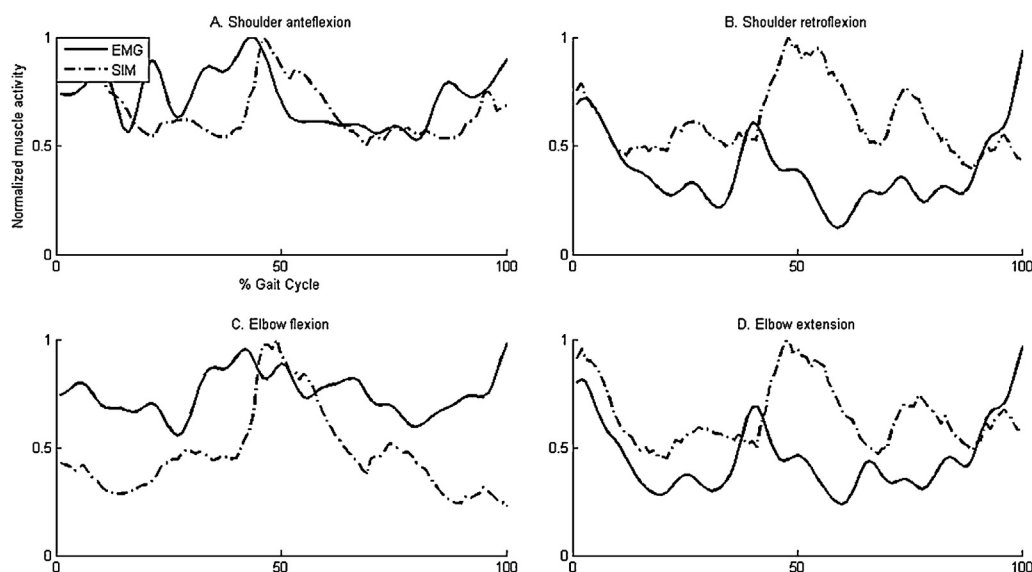


Fig. 2. Mean normalized (for gait cycle and amplitude) experimentally measured EMG activation (solid lines) and actuator excitation patterns of the simulation with arm muscle activity (ACT; dotted lines).

For the right arm, we compared the measured EMG activation patterns of the anterior deltoid and the biceps caput longum with the calculated excitation patterns of the anterior deltoid, pectoralis major, biceps caput longum and brevis and the coracobrachial actuators (panel A). For the retroflexion, we compared the measured EMG activation patterns of the posterior deltoid, triceps caput longum and latissimus dorsi muscles with calculated excitation patterns of the posterior deltoid, triceps caput longum, teres major and latissimus actuators (panel B). For elbow flexion, we compared the measured EMG activation patterns of the biceps caput longum with the excitation patterns of the biceps caput longum and breve, brachialis and brachioradialis actuators (panel C). For elbow extension, we compared the measured EMG activation patterns of the triceps caput longum with the excitation patterns of the triceps caput longum, medialis, lateralis and the anconeus actuators (panel D).

Table 1

The mean, minimal and maximal root mean square errors (RMSE) of the generalized coordinates, force output of reserve actuators and residuals of all subjects for the three different walking speeds.

The generalized coordinates are derived from the output file, pErr.sto, which contains the position errors when comparing the RRA with IK results. The results are normalized for gait cycle. Additionally, the mean, minimal and maximal RMSE in force output of the reserve actuators are shown when comparing the simulations with and the simulation without muscle activity. The results are normalized for gait cycle. Finally, the residuals (mean off all subjects), and the minimal and maximal residual values were calculated and added to the table.

RMS position errors	Pelvis tx	Pelvis ty	Pelvis tz	Pelvis tilt	Pelvis list	Pelvis rotation	Hip flex_r	Hip add_r	Hip rot_r	Knee angle_r	Ankle angle_r	Hip flex_l	Hip add_l	Hip rot_l	Knee angle_l	Ankle angle_l
Mean 0.57 m/s	3.7231	1.8834	0.9506	0.2641	0.0420	0.1318	1.7931	0.3880	0.1938	0.3444	0.2516	1.7659	0.3101	0.2427	0.3422	0.9826
Min 0.57 m/s	2.3964	0.2960	0.3081	0.1472	0.0035	0.0330	0.5040	0.1105	0.0094	0.0834	0.0160	0.5723	0.0982	0.0900	0.0462	0.2194
Max 0.57 m/s	4.8012	3.0552	1.5952	0.4579	0.0745	0.2580	3.9052	0.5639	0.4386	1.1014	0.4128	3.8242	0.6908	0.4465	1.0397	2.4301
Mean 1.11 m/s	4.5568	2.1098	1.1838	0.4467	0.0635	0.4806	3.7184	1.1427	0.6725	0.6714	0.9577	4.2650	0.5498	0.5090	0.9051	1.4481
Min 1.11 m/s	3.2112	0.1384	0.0771	0.2525	0.0129	0.0170	0.2302	0.2937	0.0054	0.0469	0.5775	0.153	0.3134	0.0945	0.0378	0.0877
Max 1.11 m/s	5.7121	3.2589	2.0830	0.6526	0.1138	1.3499	8.5808	1.8050	1.4588	2.1669	1.8141	10.1268	0.8971	0.8927	2.2113	2.9778
Mean 1.67 m/s	3.8107	1.6531	1.6214	0.5673	0.1148	0.2446	3.1689	0.6154	0.6228	0.5804	1.3604	3.0685	0.4502	0.4600	0.6583	0.9174
Min 1.67 m/s	1.4784	0.2312	0.7645	0.4564	0.0178	0.0444	0.7063	0.3544	0.0342	0.4542	1.0765	0.8188	0.0628	0.2308	0.4920	0.1624
Max 1.67 m/s	12.9696	2.9309	3.6785	1.2182	0.3626	0.6734	12.7180	1.2239	1.4449	1.4363	2.6827	12.1219	1.0071	0.8918	1.1767	1.6225

	Back tilt	Back list	Back rotation	Elv angle_r	Shoulder elv_r	Shoulder rot_l	Elbow flex_r	Pro sup_r	Elv angle_l	Shoulder elv_l	Shoulder rot_l	Elbow flex_l	Pro sup_l
Mean 0.57 m/s	0.0860	0.2129	1.7621	0.6282	0.0294	0.0162	0.0405	0.0233	0.5888	0.0254	0.0432	0.0605	0.0424
Min 0.57 m/s	0.0259	0.0033	0.6446	0.2701	0.0003	0.0023	0.0006	0.0014	0.3615	0.0136	0.0192	0.0225	0.0186
Max 0.57 m/s	0.1525	0.5845	3.5169	1.5432	0.077	0.0308	0.0701	0.0437	1.0177	0.0346	0.1026	0.0832	0.0791
Mean 1.11 m/s	0.1350	0.4167	3.1775	0.8535	0.0885	0.0337	0.1142	0.0601	3.2602	0.1036	0.0723	0.0934	0.0875
Min 1.11 m/s	0.0148	0.1179	0.8278	0.2641	0.0022	0.0145	0.0555	0.0076	0.4433	0.0235	0.0003	0.0014	0.0147
Max 1.11 m/s	0.3183	0.7288	9.8044	1.6011	0.1506	0.0660	0.1594	0.1967	12.3648	0.2664	0.1763	0.1543	0.2158
Mean 1.67 m/s	0.0607	0.4394	3.6689	1.8673	0.0581	0.0622	0.2049	0.0346	1.9049	0.0842	0.1127	0.2701	0.0790
Min 1.67 m/s	0.0152	0.1787	0.3530	0.9113	0.0066	0.0212	0.1342	0.0294	1.0521	0.0359	0.0298	0.1037	0.0069
Max 1.67 m/s	0.1417	1.3492	15.7847	4.4647	0.1777	0.1600	0.5166	0.0715	4.3041	0.2053	0.2769	0.7128	0.3210

RMSE reserve actuators in Nm	Back tilt	Back list	Back rotation	RRA residuals	Fx	Fy	Fz	Mx	My	Mz
Mean 0.57 m/s	2.5301	7.0045	13.5226	RMSE	9.0615	20.1797	2.5193	0.7386	2.5987	8.9614
Min 0.57 m/s	0.4293	0.3417	4.2568	Min res	6.8185	2.4711	0.4233	0.4193	0.7759	0.8324
Max 0.57 m/s	7.7880	24.1381	35.0171	Max res	11.2724	30.1085	4.4183	1.1914	4.7714	18.0732
Mean 1.11 m/s	4.2090	2.7531	10.85827	RMSE	10.5952	23.6999	3.3121	0.9623	1.9984	12.3519
Min 1.11 m/s	1.6035	0.3769	0.3159	Min res	4.6590	2.4445	1.3284	0.401	1.3439	0.9168
Max 1.11 m/s	7.5025	7.15817	26.2340	Max res	15.0885	32.6626	4.9553	1.8498	3.2168	22.5008
Mean 1.67 m/s	10.9988	10.8864	28.5481	RMSE	7.8249	21.7869	4.1316	0.9816	4.0235	5.2829
Min 1.67 m/s	0.6044	3.4485	18.4357	Min res	1.1078	8.0068	3.2524	0.6116	1.0553	0.3053
Max 1.67 m/s	25.3010	13.7890	42.9693	Max res	16.727	31.5840	6.1396	1.3483	7.1674	8.8511

passive muscle characteristics control arm swing. This allowed us to assess the role muscle activity plays in arm swing during human walking.

In line with literature [3,8,10,11,25], our results showed that arm swing during walking is partially arising from passive dynamics. However, a significant decrease of arm swing amplitude when no muscle activity is present was found, confirming that passive dynamics alone cannot induce sufficient swing amplitude. Moreover, when muscle activity was absent, the arm patterns changed to a more in-phase pattern.

The need to add actively control to passive induced arms motion confirms previous work by Elftman [26] and others that used pendulum-like models [8–10] to investigate arm swing kinematics. All these studies have reported a passive and active component in arm swing during walking. However, this study is the first to use a complex musculoskeletal model that can account for the experimentally measured lower limb, upper limb and torso kinematics, thereby accounting for the complex segmental coupling that determines arm swing. The musculoskeletal model incorporates a Hill-type muscle model [27] that results in a plausible physiology-based description of the active, but even

more importantly passive muscle characteristics, when analyzing the passive component of arm swing.

Our study furthers the insights gained from previous simulation studies: our results confirm that activity of arm muscles regulates relative phase (and therefore controls arm swing direction, in line with what Jackson et al. [8] found), but that muscle activity is also needed to increase arm swing amplitude, which confirms what Kubo et al. [10] hypothesized.

Surprisingly, for the simulations without muscle excitations (PAS) arm swing amplitude was similar for the three different walking speeds. This suggests that the passive dynamics [11] (e.g., muscles and accelerations of neighboring segments) were comparable and that walking speed had little influence. The same is true for the similarity in relative phase at walking speeds 1.11 m/s and 1.67 m/s. This is partially in agreement with the finding by Bruijn et al. [1] that the thorax has a similar timing of rotation at all speeds, with only a minor decrease in rotational amplitude. Since stride times reduce with increasing walking speed (which was also seen in our subjects), this will result in an equal increase in accelerations and decelerations of the thorax with increasing walking speed [28]. Therefore, net thorax acceleration is similar for

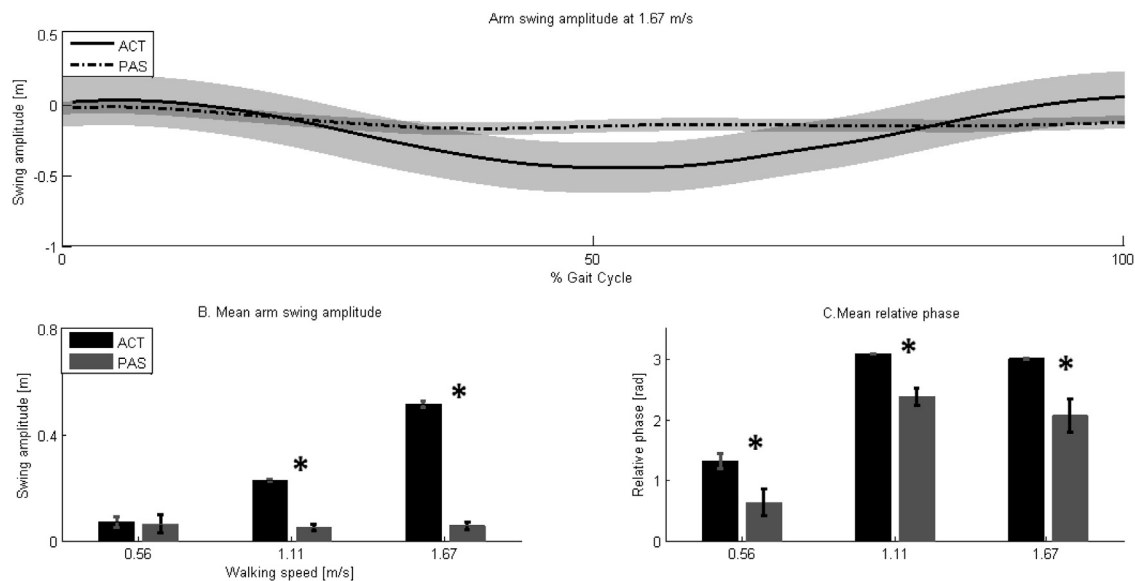


Fig. 3. Differences in arm swing amplitude and relative phase for the two simulations (ACT & PAS).

The first panel (A) shows the mean normalized (for gait cycle) arm swing amplitude of all subjects for the simulation with (solid line) and without (dotted line) arm muscle activity at walking speed 1.67 m/s with their standard deviations (grey band). The second panel shows the mean swing amplitude (B) at the three walking speeds (0.56 m/s, 1.11 m/s and 1.67 m/s) for the ACT (black) and PAS (grey) data (mean \pm SD). The last panel (C) displays the mean relative phase of the arm swing (difference between de right and left arm calculated in rad.) for the ACT (light grey) and PAS (dark grey) data (mean \pm SD). Significant differences ($p < 0.05$) between the two simulations (ACT & PAS) are indicated with a*.

all three walking speeds and this is reflected in the unaffected arm swing amplitude.

One of the limitations of this study is that it focuses mainly on the 'how' of arm swing. We acknowledge that the 'why' arm swing is controlled the way it is, is an important part of understanding arm swing in human walking as well. As was also mentioned in the introduction, the active component in arm swing is essential in maintaining the out-of-phase swing pattern. As a result, and perhaps counter-intuitively, the presence of active muscle control seems crucial in minimizing energy expenditure as confirmed in previous research [2,4,5,15,29].

Further, inherently to the use of the CMC tool in OpenSim, reserve actuators were added (torques) to control the DOF of the trunk and lower limb joints (not for the upper limbs). As such, the kinematics could still be accurately tracked even when insufficient muscle forces were generated. However, we visually verified that the magnitude of the reserve actuator contribution only minimally changed between the two simulations (ACT & PAS, Table 1).

Additionally, we were not able to collect EMG data from all shoulder and upper arm muscles, therefore, we could not validate each individual muscle, but were only able to compare EMG data with the grouped synergistic actuators data.

Finally, other than the passive forces provided by the actuators, we did not account for passive forces from ligaments, joint capsule or other soft tissues in our model. These passive forces might cause a further decrease in the magnitude of the passive arm swing due to an increase in stiffness around the joint. However, we believe that the influence of these passive forces is minimal, since arm swing kinematics during walking do not reach the joint positions in which the ligaments provide substantial resistance.

5. Conclusion

We conclude that there is a passive component in arm swing during walking, but with reduced arm swing amplitude and a more in-phase pattern when compared to walking with arm muscle

activity. Therefore, muscle activity is needed to increase arm swing amplitude and modify relative phase during human walking to induce out-phase swinging.

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Conflicts of interest

There were no conflicts of interest.

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